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Diagnosis of hepatic hydrothorax in the absence of ascites by intraperitoneal injection of 99m-Tc-Fluor colloid

Sir,

Hepatic hydrothorax affects approximately 5% of patients with cirrhosis of the liver.¹ Although usually associated with ascites, some patients show no clinically detectable intraperitoneal fluid², and a few cases with negative abdominal ultrasonographic examination have also been reported.³

The pathogenesis of hepatic hydrothorax has been controversial. Some studies have demonstrated a transdiaphragmatic flow of ascitic fluid to the pleural space through communications between peritoneum and pleura, which have also been observed at necropsy. We report a cirrhotic patient who developed hepatic hydrothorax. The administration of colloid Fluor-Sn-Tc99m into the peritoneal cavity demonstrated a peritoneal-pleural communication when there was no ultrasonographic detectable ascites.

A 66 year-old male with alcoholic cirrhosis and previous ascites and right pleural effusion was admitted in September 1989 with a massive right pleural effusion, but no clinically detectable ascites. The serum albumin level was 34 g/l and the pleural fluid examination indicated a transudate. Abdominal ultrasonography showed no evidence of ascites. A total of 370 MBq of Fluor-Sn-Tc99m was injected into the peritoneal cavity. Thorax and abdomen were scanned and images obtained at 15 min and 5 h showed a continuous and progressive flow of the radiopharmaceutical into the pleural cavity. A rectilinear hyperactivity over the right hemidiaphragm, suggesting the existence of a communication could also be

observed. A chemical pleurodesis with tetracycline gave only a transitory benefit. Two weeks after this the effusion recurred and the patient died 3 months later. The necropsy showed mild ascites and a large pleural effusion. However, a detailed examination of the right hemidiaphragm failed to evidence any defect.

This case report demonstrates the natural evolution of hepatic hydrothorax in several respects. First, the patient developed a massive pleural effusion with a progressive reduction in the amount of peritoneal fluid which could not eventually be demonstrated by ultrasonography as reported previously.³ Thus absence of ascites cannot exclude the cirrhotic aetiology of a pleural effusion. Second, immediately after each thoracentesis, we noticed a rapid reaccumulation of pleural effusion. This can be explained by transdiaphragmatic flow of fluid, due to the negative intrapleural pressure.

Thoraco-abdominal scintigraphy with intraperitoneal administration of Tc99m colloid has been useful in the diagnosis of hepatic hydrothorax⁴ even in the absence of clinical ascites.³ The case reported is the first to our knowledge in which radionuclide imaging has been performed with ultrasonographic proven absence of ascites.

With respect to the treatment of hepatic hydrothorax the combination of chemical pleurodesis and peritoneal shunt should probably be used⁵ as conservative measures usually fail.

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